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Introduction

Neurosonography is an important test in the diagnosis of hemorrhage and other acquired and congenital brain pathology of the newborn. Premature neonates are especially at risk for intracranial problems, and it is important for the sonographer to have a thorough knowledge of the normal anatomy and sonographic appearance of the neonatal brain. Many sonographers learn neonatal brain imaging in school and have no further education on the subject during their careers. As continuing education in ultrasound is necessary to maintain proficiency and reduce the chance of missing pathology, the purpose of this article is to discuss the normal sonographic appearance of the neonatal brain as well as some common pathologic conditions.

Despite the advances in computed tomography (CT) and magnetic resonance imaging (MRI), ultrasound (US) is the most commonly used modality for examining the newborn brain.

Ultrasound is still the only modality able to image the brain at the bedside, which can be vitally important in the case of the critically ill infant. Whereas CT and MRI require sedation for optimal imaging, US can be done without incurring the risks associated with sedation. Also of benefit to the newborn is that ultrasound is easily reproducible and does not produce any ionizing radiation(1). Of the three aforementioned modalities, however, ultrasound is by far the most operator-dependent. While 3D volume acquisition can reduce or eliminate most of the interoperator variability, most neonatal neurosonography is still performed with freehand 2D imaging(2).

Anatomy

As with all areas of ultrasound, thorough knowledge of the normal anatomic structures is essential to the sonographer. The brain can be divided into the cerebrum, brain stem and the cerebellum. The cerebrum, or upper portion of the brain, is composed of four lobes: the frontal lobe, parietal lobe, temporal lobe and occipital lobe (Figure 1). These lobes are separated by fissures, which are infoldings of the brain tissue. Fissures important to the sonographer include the sagittal fissure, which separates the left and right parietal lobes and the Sylvian fissure, which separates the parietal and temporal lobes. The falx cerebri, commonly called the “falx,” is an infolding of dura mater separating the left half of the cerebrum from the right(3).

It is important to be able to identify the midline structures of the neonatal brain because not only are there important landmarks that divide the left side from the right side, but knowledge of what the structures are supposed to look like will help identify midline abnormalities. Figure 2 demonstrates a complete sagittal view of the midline. Located in the center of the image in the midline is the cavum septum pellucidum (Figure 3), a fluid-filled

structure commonly seen on prenatal ultrasound. Extending posteriorly from the cavum septum pellucidum is the cavum vergae, which begins to disappear at approximately six months gestation. The echogenic structures in the midline that form the choroid plexus and cerebellar vermis have an appearance that, with one's imagination, looks like a woman in Victorian-era dress, the "lady in the dress" sign (Figure 4). When the "lady in the dress" is present in the image inferior to the cavum septum pellucidum, the entire midline plane is being visualized. Just superior to the cavum septum pellucidum is the corpus callosum, a medium-gray level bundle of never fibers that provides the communication between the cerebral hemispheres. As demonstrated in Figure 3, inferior to the cavum is the third ventricle, thalamus and brain stem. Located posteroinferiorly in this view is the echogenic vermis of the cerebellum(3).

The purpose of the ventricular system of the brain is the distribution of cerebrospinal fluid (CSF). The ventricular system is comprised of the paired lateral ventricles, the 3rd ventricle and the 4th ventricle. The left and right lateral ventricles drain into the 3rd ventricle via the Foramen of Monro, the third ventricle drains into the fourth ventricle via the aqueduct of Sylvius and the 4th ventricle drains into the subarachnoid space via the foramina of Luschka & Magendie. Blockage at any point along this path results in a buildup of CSF and can lead to hydrocephalus.

Within the lateral ventricles is the bulk of the choroid plexus. This brightly echogenic structure exists in the lateral ventricles, third and fourth ventricle. However, the choroid plexus only exists in certain parts of these structures, which is significant because the presence of echogenic materials outside of these specific regions may indicate blood with in the intraventricular system. In the lateral ventricles, the bulk of the choroid plexus is seen in the trigone, or atrium. Choroid plexus can also be found in the roof of the third and fourth ventricles

as well the temporal horns of the lateral ventricles. It is not uncommon, especially in premature neonates, to see a choroid with a “lumpy bumpy” appearance in the atrium of the lateral ventricle. This is a normal variant, however if there is concern for hemorrhage then the sonographer needs to be diligent in ruling out the presence of blood in the frontal (anterior) or occipital (posterior) horns, where no choroid is normally present.

Inferior to the cavum septum pellucidum but lateral to the midline are two homogenous, hypoechoic, round structures: the thalamus and the head of the caudate nucleus. The junction where these two structures meet is called the caudo-thalamic notch (CTN), or groove. The choroid plexus extends from the atrium of the lateral ventricle anteriorly, tapering to a point at the level of the CTN (Figure 5). In the region of the CTN is the germinal matrix, a hypervascular endothelial lining which lies deep to the ependyma. The germinal matrix is a friable collection of blood vessels that disappears as the fetus grows in utero. Although this lining is not visualized with ultrasound, the area where the germinal matrix resides is examined extensively because of the risk of bleeding in this area. Initially lining the entire ventricular system, the germinal matrix regresses until it lies only within the CTN, at approximately 24 weeks. By 32 weeks, the risk for bleeding in the germinal matrix is greatly reduced, and disappears completely by 40 weeks(3). The appearance of the brain parenchyma varies with gestational age, as well. The brain appears smooth, without sulci or gyri, up to about 22 weeks, and then continues to mature until term(4), as demonstrated in Figure 6.

In the posterior brain lies the cerebellum, which has an echogenic central portion termed the vermis. The cerebellum is responsible for motor control and coordinated movement, and lies posterior to a tent-like structure called the tentorium. Structures that lie in this region are said to

be “infratentorial.” Anterior to the cerebellum lies the fourth ventricle, which appears as a triangular-shaped, echo-free area(4).

A small amount of extraaxial fluid may normally be seen surrounding the brain, and is typically more prominent in the premature neonate. However, brain atrophy, infection, communicating hydrocephalus and hemorrhage may be responsible for abnormal extraaxial collections(4).

Scanning – Technique and Protocol

In preparation for performing an ultrasound of the neonatal brain, it is essential that proper antiseptic precautions be undertaken due to the poor immune system of neonates, especially those born premature. Proper hand washing of the sonographer and disinfection of the transducer are essential in order to reduce transmission of infectious agents to the immunocompromised newborn(5). It is also important to maintain the neonate’s body temperature, as the newborn is susceptible to rapid heat loss, so if at all possible, keep the patient covered (if scanning in the isolette) or under a warming lamp. If single-use gel packets are used, they may be passively warmed up by keeping them in a shirt pocket or on a warm part of the ultrasound machine.

Ultrasound of the neonatal brain is usually performed with a small footprint, high-frequency phased array transducer with either a sector, vector or small field-of-view curvilinear image pattern. Transducer frequency depends on the age of the patient; while a 7-10 MHz may be suitable on a preterm neonate, a 5-7 MHz may be more suitable on a child that is > 3 months of age. Proper depth of view is important to ensure that no pathology is missed in the posterior portion of the brain, and the use of multiple focal zones will improve lateral resolution. To

ensure proper depth, the posterior/inferior cranial bones should be visualized on every non-magnified image (Figure 7) (6).

As with most ultrasound studies, it benefits the sonographer to consider the use of alternate transducers to obtain a better image if necessary, and neonatal ultrasound is no exception. For example, the use of a high-frequency linear transducer as an adjunct to the small footprint transducer can be used to image the super sagittal sinus to rule out thrombosis, to image the cerebral cortex and to evaluate the meninges for inflammation or subdural blood (7).

Most neurosonography imaging is performed via the anterior fontanel, although other windows may be used to visualize structures from different vantage points. The most commonly used alternative windows include the posterior and mastoid fontanels (8). The posterior fontanel views are often part of a standard protocol to ensure that no pathology is missed in the occipital horns and infratentorial structures such as the cerebellum and surrounding anatomy.

Imaging should include both sagittal and coronal planes, supplemented with axial views (via the mastoid fontanel) as necessary. A protocol is included below, although note that these are only the *minimum* views that should be obtained. In the presence of pathology, of course, additional views must be documented.

In the sagittal plane, the following images should be documented (Figure 8 a-f):

- True sagittal midline view (the “lady in the dress” view)
- Oblique parasagittal image of the CTN
- Lateral ventricles including frontal, temporal and occipital horns
- Images of the brain tissue lateral to the ventricles (to include the Sylvian fissure) to examine middle cerebral artery (MCA) pulsations

Imaging in the coronal plane should include these views (Figures 9 a-f):

- Anterior-most view including the orbits
- Frontal horns
- Frontal horns (with and without measurements) at the level of the third ventricle where the choroid is seen in the roof of the third ventricle (see Figure 10 for magnified view)
- Atria and occipital horns of the lateral ventricles
- Posterior brain tissue

While the lateral ventricles may lie mostly parallel to the falx, they often require a slight "twist" of the transducer from midline to obtain a parasagittal-oblique view of the ventricle in its entirety, as seen in Figure 8d. The CTN view is obtained by starting in the true sagittal plane and slightly rotating the heel of the transducer laterally (Figure 11). The choroid plexus should be seen coursing in an anterior direction cephalad to the thalamus, tapering to a point in the depression between the thalamus and caudate nucleus. The choroid should always be seen to taper in this region; any echogenic material in the CTN should be considered a possible bleed. The remained of the lateral ventricles are imaged with the transducer in the parasagittal plane.

Careful examination should be performed of the frontal, temporal and occipital horns via the anterior fontanel. The ventricles should be evaluated for size and shape as well as for the presence of blood. Remember that there should be no echogenic material seen in the frontal horns, occipital horns, or dependent portion of the temporal horns. Any echogenic material in the wrong place should be considered a bleed until proven otherwise(1). While most neonatal brain imaging is performed via the anterior fontanel, evaluation of the occipital horns should be performed via the posterior fontanel as well, to ensure proper visualization of the occipital horn, as demonstrated in Figure 12 (8).

Intracranial Hemorrhage

One of the main indications for ultrasound of the neonatal brain is to evaluate the ventricular system for the evidence of intracranial hemorrhage (ICH), colloquially referred to as a "bleed." In a grading system first described by Papile (9) and later modified by Volpe (10), intraventricular hemorrhages (IVH), are commonly labeled as I, II and III. The grade I bleed, also referred to as a subependymal hemorrhage (SEH) or germinal matrix hemorrhage (GMH), is the mildest of the bleeds and typically has no lasting neurological sequelae (11). A bleed is considered to be a Grade I when the blood is confined to the region of the CTN. Remember that the choroid plexus normally tapers to a point in the CTN, which is the location of the germinal matrix after 23+ weeks gestation. With a Grade I bleed, instead of tapering, the choroid will appear bulbous as it dives anteriorly into the CTN (Figure 13). Grade I bleeds may vary in size from very small to several centimeters, but are always confined to the CTN.

A Grade II bleed is a bleed that has escaped the confines of the CTN and is now freely intraventricular. Blood may be seen anywhere in the ventricular system, including the frontal horns and the occipital horns, but the ventricles remain normal in size, as seen in Figure 14 (1). It is important to use the posterior fontanel routinely to rule out blood in the occipital horns, as the anterior fontanel view limits visualization of the occipital horns due to artifact related to increased depth between the transducer and the infratentorial structures (8).

For a bleed to be considered a Grade III, ventricular dilatation will be present in addition to intraventricular blood (Figure 15). Whereas Grade I and II bleeds are considered to have no long lasting impact on neurological outcome, Grade III bleeds have a significantly higher

mortality rate and higher neurological impact due to hydrocephalus and increased pressure on the brain tissue (11).

The so-called Grade IV bleed is an intraparenchymal hemorrhage (IPH) that may or may not be associated with an intraventricular bleed. It used to be thought that Grade IV bleeds were a progression from Grade III bleeds, but the current literature recognizes that IPH may occur on its own and is of a different etiology compared to IVH (10). Grade IV bleeds commonly appear as an echogenic mass (or masses) in the frontal or parietal lobes, as seen in Figure 16. One of the chief concerns with the Grade IV bleed is the loss of brain tissue that results from degeneration of the resulting necrotic area. Regardless of the cause, the neonate with a Grade IV bleed is at very high risk for adverse neurological outcome (11). As the blood is resorbed, the necrotic brain matter may connect to the ventricles. The resultant porencephalic cyst is lost brain matter and carries with it a very poor neurologic outcome (Figure 17) (12).

Hydrocephalus/Ventriculomegaly

Ventriculomegaly, or dilatation of the ventricles, has several etiologies. It may be associated with congenital anomalies, associated with either increased production or decreased absorption of CSF, or seen in conjunction with the sequelae of intracranial hemorrhage. Clinically, hydrocephalus may present with a prominent ("bulging") anterior fontanel, rapid head circumference growth and separated cranial sutures (13). In neonates who survive to infancy, hydrocephalus is the most common congenital malformation. Although the terms "hydrocephalus" and "ventriculomegaly" are often used interchangeably, the term "hydrocephalus" is used in by some authors when there is an obstructive cause for the dilatation of the ventricles (4) or there is increased pressure (14).

Hydrocephalus can be divided into communicating and non-communicating. Non-communicating hydrocephalus is when the dilatation is the result of blockage from within the ventricular system, and communicating hydrocephalus is the result of either decreased absorption or blockage from outside the ventricular system (4). Neonatal infection, intracranial masses and abnormal vascular processes are other possible causes of hydrocephalus. Hydrocephalus can be associated with poor outcome, depending on the etiology. Multiple studies have demonstrated normal mentation occurring anywhere between 15-90% of neonates (15),(16),(17),(18).

After intracranial hemorrhage, atrophy of the brain tissue may cause post-hemorrhagic ventricular dilatation (hydrocephalus *ex vacuo*) (14). It is important to note that post-hemorrhage hydrocephalus does not show clinical symptoms right away, and it can take one to three weeks after the hemorrhagic event for clinical signs to manifest. For this reason serial ultrasound is important in the diagnosis of hemorrhage-associated hydrocephalus (19). Ventriculomegaly is diagnosed by measurement of the frontal horns at the level of the third ventricle. At Thomas Jefferson University Hospital, a measurement of $\geq$ 4mm is considered "dilated." Dilatation of the third ventricle may reveal a tissue bridge between the two lobes of the thalamus called the massa intermedia, not to be confused with intraventricular clot (Figure 18).

Periventricular Leukomalacia

In cases of hypoxia, necrosis of the periventricular white matter may occur, termed periventricular leukomalacia (PVL). Sonographically, PVL initially appears as an area of highly echogenic tissue in the parietal lobe adjacent to the lateral ventricles or in the frontal lobes (Figure 19). Diagnosis relies on the echogenicity of the periventricular brain; if the parenchyma adjacent to the lateral ventricles appears more echogenic than the choroid plexus, PVL must be

considered (4).The most significant sequela of PVL is cystic change of the affected brain matter resulting in a “Swiss-cheese” like appearance of the parenchyma. This cystic change may occur days to weeks after the initial insult. Severe PVL may eventually evolve into cystic encephalomalacia and porencephaly (Figure 20), which are may lead to cognitive and seizure disorders (19).

Congenital Anomalies of the Neonatal Brain

A variety of congenital anomalies of varying severity may occur in the brain. These anomalies may range from mild, with no adverse neurological outcomes (such as a lobulated choroid plexus), to severe, including hydranencephaly and alobar holoprosencephaly. While there are many texts on neurosonography detailing all of the potential intracranial anomalies, this article will highlight the more commonly seen pathologies.

Abnormal brain structure may be seen as the sequela of a spinal condition such as myelomeningocele and other open neural tube defects. With open spinal defects, the spinal cord is displaced through a defect, typically located at the base of the spine. This downward displacement of the spinal cord pulls the brain through the only available opening, the foramen magnum at the base of the skull. The cerebellum and brainstem are pulled downward, obliterating the cisterna magna and causing obstruction of the cerebrospinal fluid flow. While the frontal horns appear small, the posterior horns of the lateral ventricles are typically enlarged and tear-dropped shaped, an appearance termed colpocephaly (20). There are several midline brain defects that may visualized in neonatal brain imaging. One type that occurs is called agenesis of the corpus callosum (ACC). In embryologic development, the anterior corpus callosum develops first, followed by the posterior components (1). Agenesis of the corpus callosum may be partial

or complete, but if partial typically involves absence of the posterior portion related to the later development of this portion of the corpus callosum. It is important to note if there are other associated defects or if the ACC is an isolated event. Postnatal MRI is usually performed to confirm the type of ACC, whether partial or complete, and to look for other defects as well (1). In the case of isolated ACC there is usually a good prognosis, although affected children have been demonstrated to have seizures or developmental delay (21). The sonographic features that suggest ACC include a high-riding third ventricle and failure to visualize the corpus callosum in its usual location. As demonstrated in Figure 21, the frontal horns appear to be markedly separated, and the lateral ventricles run parallel to each other. The occipital horns may appear tear-drop shaped (colpocephaly), and the sulci/gyri run perpendicular to the third ventricle instead of parallel to it, causing the so-called "sunburst" sign (20).

One of the most dramatic defects of the midline that can occur is holoprosencephaly. Holprosencephaly is divided into three types (in decreasing order of severity): alobar, semilobar and lobar. With alobar holoprosencephaly there are severe intracranial, facial and midline defects. Alobar holoprosencephaly, which is almost uniformly lethal even without chromosomal abnormalities, is also often associated with lethal genetic disorders such as trisomy 13, trisomy 18 and triploidy (22). The striking intracranial findings of alobar holoprosencephaly typically include a dilated single, central monoventricle; a thin, peripherally located cerebral cortex; fused thalami and absent falx cerebri. Facial features often include a orbit anomalies, varying from a more mild hypotelorism to a single orbit containing one or two eyeballs; a superiorly-located proboscis and severe cleft lip/palate defects (23).

Semilobar holoprosencephaly is less severe than the alobar form, but shares some of the midline characteristics. With semilobar, the thalami are only partially fused and a partial falx

may be seen. There may be mild facial abnormalities or there may be none at all. Lobar holoprosencephaly, the mildest form, may present as ACC and hypoplasia of the optic nerve. With lobar holoprosencephaly, the falx cerebri is complete or nearly complete, and there are two cerebral hemispheres (23). The prognosis is better for the lobar form than with the other forms of holoprosencephaly, although there may still be severe mental retardation. Facial anomalies, if present, are usually mild (1).

Anomalies of the posterior brain, such as Dandy Walker and Dandy Walker variant are other brain anomalies that may be seen with neonatal intracranial ultrasound. The Dandy Walker complex is visualized as a posterior fossa cyst that communicates with the fourth ventricle. The cerebellar vermis is either hypoplastic or absent, and there may be secondary third or lateral ventricle dilatation caused by atresia of the foramina of Luschka and Magendie. With Dandy Walker variant, a normal or hypoplastic cerebellar vermis is present, and instead of a cyst there is an enlarged cisterna magna that communicates with the fourth ventricle. Either condition may be associated with other structural defects and/or chromosomal anomalies (Figure 22) (24).

Neonatal Infection

Transmission of maternal infection to the neonate is another condition encountered by the neonatal sonographer, and a serious concern for the patient. Infectious diseases that may be transmitted via the placental or birth canal are toxoplasmosis, rubella, cytomegalovirus (CMV), and herpes, the so-called TORCH infections. The hallmark of intracranial infection is calcification within the brain of the affected neonate, although cystic encephalomalacia may also be seen, resulting in neurodevelopmental delay (25).

Three-Dimensional Ultrasound

With advances in three-dimensional US (3DUS), it is now possible to acquire a volume set of images and visualize the brain in multiple planes via digital reformatting. With 3DUS, there is a decrease in the sonographer acquisition time compared to 2D imaging, although the time it takes for the physician to interpret the images increases due to the potential multiple planes that can be obtained. Figure 23 demonstrates 2D slices of the neonatal brain obtained with volume imaging. After capturing the volume it is sliced into axial, coronal and/or sagittal planes as needed. The decrease in acquisition time with 3DUS is important to consider due to the fragile health of the preterm neonate. Very low-birthweight neonates are less tolerant of being touched, and there is the added risk that tubes or lines may be displaced with increased scanning time. Another advantage of 3DUS is that it removes the interoperator variability with obtaining the images, as well as decreases the chance that pathology may be missed. The chief downside with modern 3DUS is that the 3D reconstructions are often of less quality then the standard 2D images. With improvements in computer reconstruction, however, it is possible that 3DUS will reign superior to 2D imaging for imaging of the neonatal brain (26). As with all 3D imaging, though, a good 3D study is not possible without a good 2D image. If there is an inadequate window or much artifact, it will not be likely that an acceptable 3D can be performed.

As the quality of the reconstructions of 3DUS advances, it makes it easier to compare US images with comparable CT and MRI views. Three-dimensional US also has the added benefit of educational and training opportunities, as any study can be "virtually rescanned" at a work station long after the scan is completed (27).

Doppler in Neurosonology

As with most areas of ultrasound, Doppler may be used to evaluate for flow patterns and velocities. Doppler, although not used routinely in many centers, may be used to evaluate the perfusion to the brain via the anterior cerebral artery (ACA) and middle cerebral artery (MCA). Velocity information is used in conjunction with resistive indices (RI), as a change in the RI may be a predictor for prolonged asphyxia with ICH and/or cerebral edema (28). The resistive index, which is a marker of the impediment to flow of a vessel bed, is susceptible to extracranial influences such as a patent ductus arteriosis (PDA) and therefore may vary. At least one author has developed charts for normal resistive indices of the MCA, ACA and internal carotid arteries (29), and another author is using RI information along with graded compression of the anterior fontanel to predict which neonates would benefit from shunting in hydrocephalus (30).

Conclusion

Unlike other areas of sonography (e.g., abdomen and ob/gyn), neurosonography is not a common study in most non-pediatric institutions. Many sonographers learn the topic in school and then work in a hospital or outpatient center that doesn't have a neonatal unit. When the time does come to perform these exams, it is often learned or re-learned via on the job training. As technology and technique are constantly changing, all areas of ultrasound require continuous education and training; it truly is a field in which we are all students regardless of the number of years behind us. Neurosonography is an important tool in the diagnosis of intracranial problems in the newborn, and one without the problems of sedation or ionizing radiation. The use of neurosonography allows for the prediction of neurological development and outcome in this high-risk population, and it is critical for the sonographer to stay up to date with this ever-changing field.

Captions

Figure 1 – Long-axis view of the brain showing the four lobes of the cerebrum.

Figure 2 – True sagittal midline.

Figure 3 – Midline structures of the neonatal brain. Fat white arrows: corpus callosum. Thin white arrow: third ventricle. Black arrow: thalamus. Chevrons: vermis of the cerebellum. Arrow head: fourth ventricle.

Figure 4 – (a) Midline brain showing the echogenic structures representing the choroid in the third ventricle and cerebellar vermis. (b) and (c) showing the "lady in the dress" sign. Demonstration of the corpus callosum and the "lady in the dress" ensures a completely true-sagittal midline view.

Figure 5 – Para-sagittal view of the caudo-thalamic notch. The choroid plexus tapers in this region and should not increase in size anteriorly.

Figure 6 – (a) neonatal brain at 26 weeks. Note the lack of defined sulci/gyri. (b) neonatal brain at 37 weeks gestational age. Note the pronounced sulci/gyri seen with maturity.

Figure 7 – (a) posterior structures are being cut off, as evidenced by lack of visualization of the inferior cranial bones. In image (b), the cranial bone is seen in its entirety.

Figure 8 (a - f) – Progressive sagittal sections through the neonatal brain. This is the minimum that needs to be documented in a normal brain.

Figure 9 (a - f) – Progressive coronal sections through the neonatal brain. This is the minimum that needs to be documented in a normal brain.

Figure 10 – coronal, magnified view of measurement of the frontal horns of the lateral ventricles. The frontal horns are measured at the level of the choroid plexus as it resides in the 3rd ventricle (arrow).

Figure 11 – a) Illustration of the transducer oriented in sagittal plane for obtaining midline image. b) A slight rotation of the transducer from the midline is required in order to obtain a caudo-thalamic notch view

Figure 12 – Occipital horn as seen through the posterior fontanel.

Figure 13 (a – c) – Grade I hemorrhage, or sub-ependymal hemorrhage. Notice that the bleed is confined to the CTN and that the choroid does not taper normally as it travels anteriorly.

Figure 14 (a – c) – Grade II hemorrhage. Note clot extension into occipital horn (arrow).

Figure 15 (a – c) – Grade III hemorrhage. There is ventricular dilatation and clot throughout ventricular system.

Figure 16 (a – d) – Grade IV, or intraparenchymal hemorrhage. Note the scattered echogenic areas consistent with parenchymal blood. There is also significant intraventricular blood, as well.

Figure 17 – Grade IV hemorrhage with porencephalic cysts bilaterally. There is an increased amount of extraaxial fluid, as well, consistent with atrophy of the brain.

Figure 18 – Dilatation of the third ventricle revealing the massa intermedia, the tissue bridge between the two lobes of the thalamus.

Figure 19 – (a) early PVL. Note the areas of increased echogenicity adjacent to the lateral ventricles. (b) Several weeks later, cystic change has occurred in the parenchyma.

Figure 20 – severe cystic encephalomalacia, related to early prematurity and PVL.

Figure 21 – Agenesis of the corpus callosum. (a) Widely spaced anterior horns (b) Absence of normal midline structures. The corpus callosum and cavum septum pellucidum are absent. (c) Tear-dropped shaped occipital horns (colpocephaly)

Figure 22 – (a) coronal ultrasound of Dandy-Walker variant. Note the splayed cerebellar tonsils and absence of the echogenic central vermis. (b) Coronal magnetic resonance (MRI) scan of the same patient.

Figure 23 – Three-dimensional volume imaging of the neonatal brain. (Images courtesy of Philips Healthcare, Bothell, WA).

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Figure 1

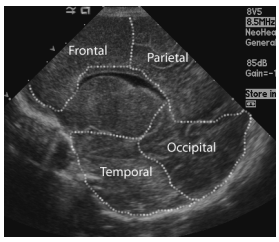
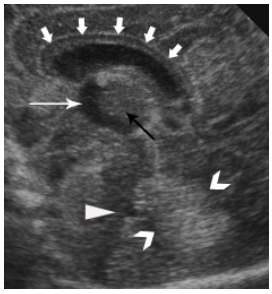


Figure 2

Figure 3



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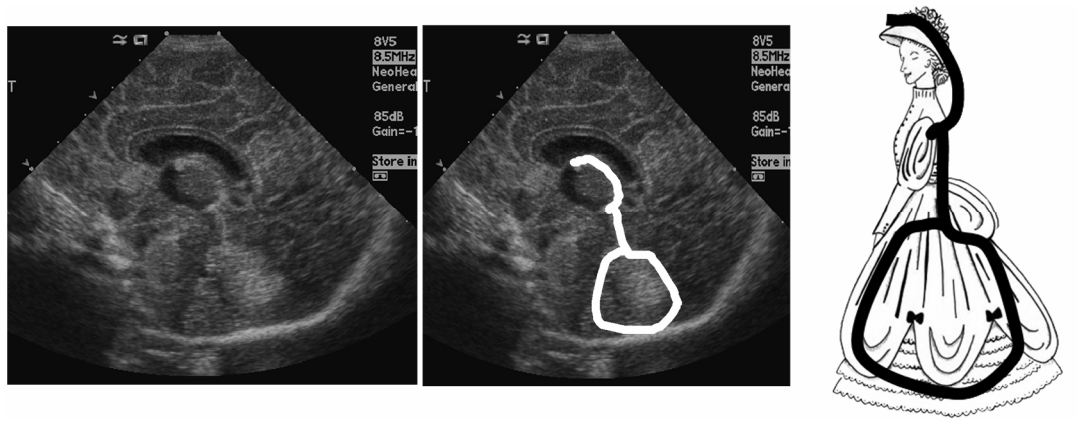

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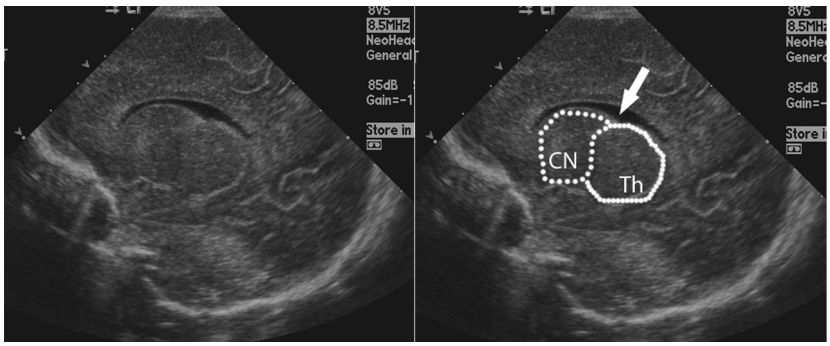

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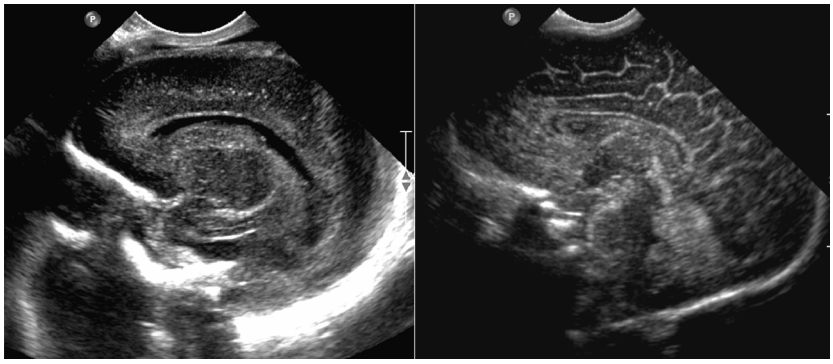

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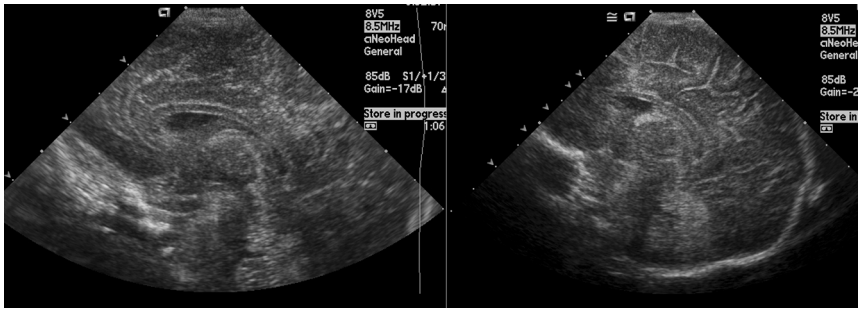

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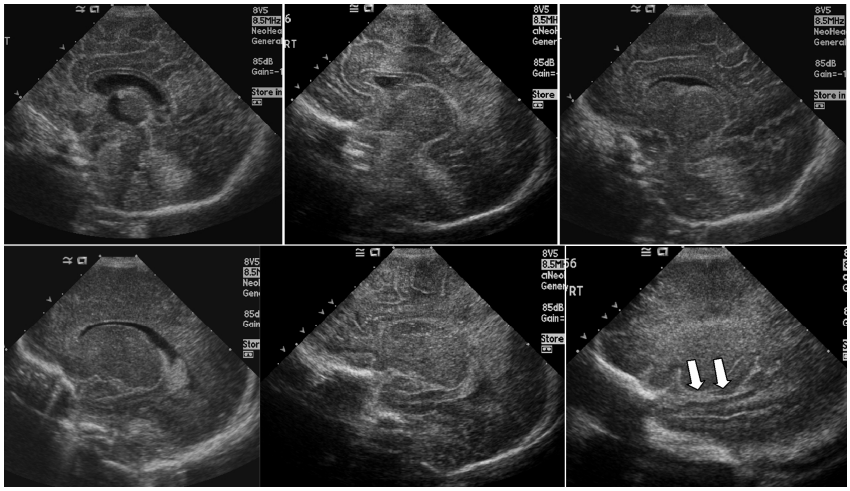

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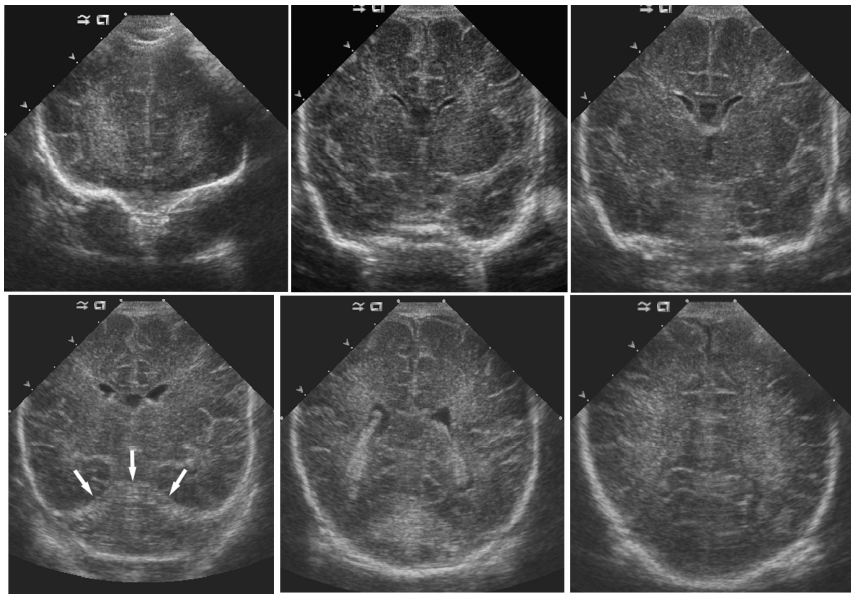

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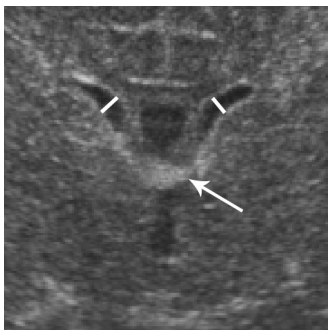

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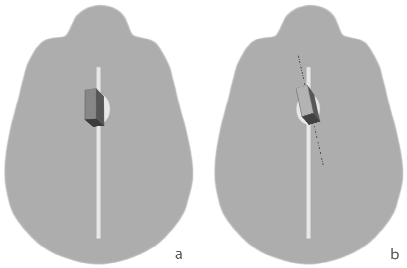

Figure 12

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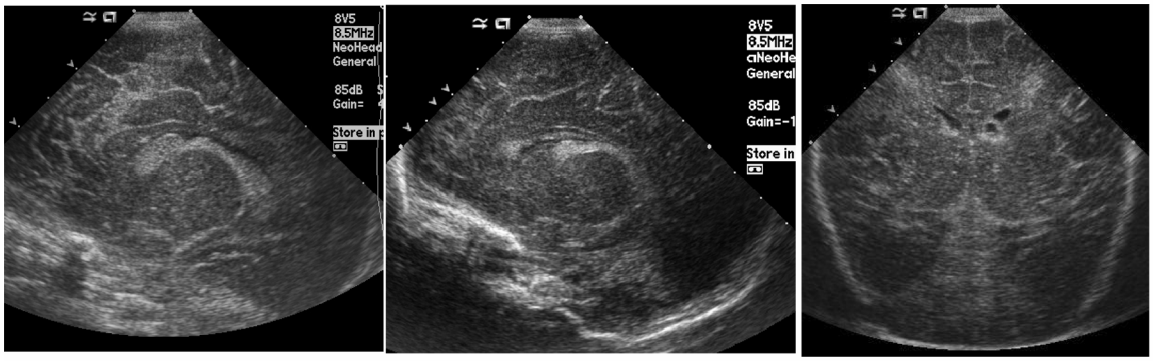

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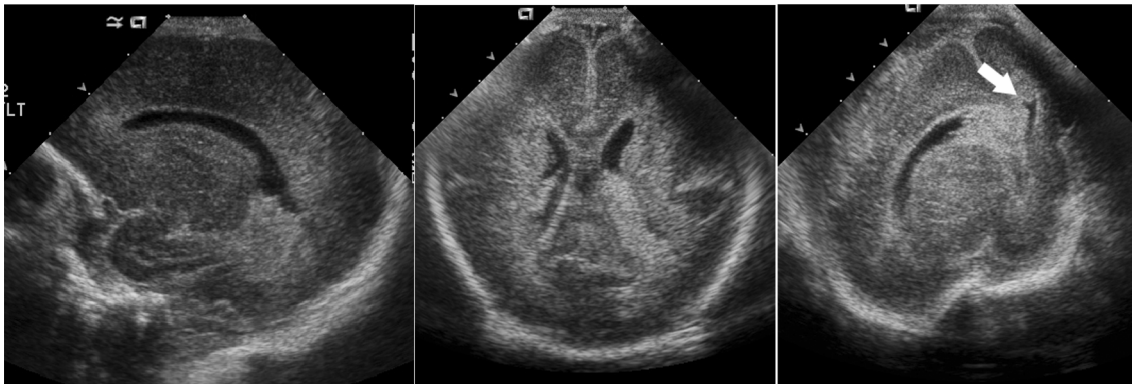

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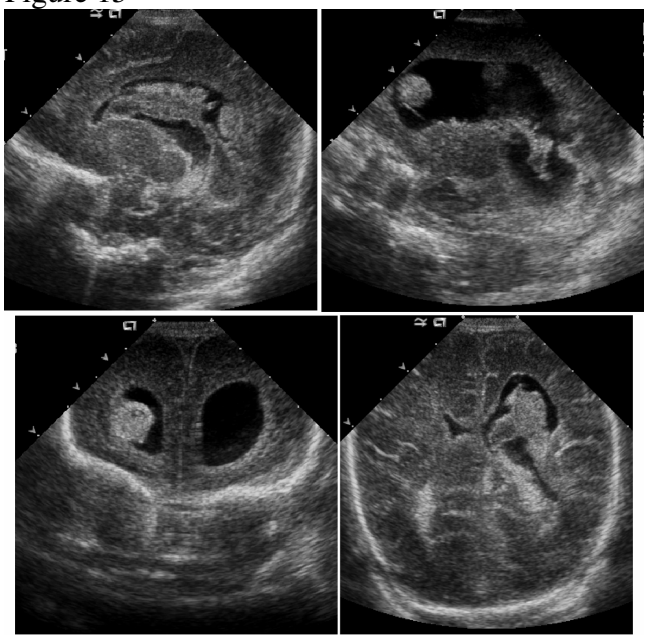

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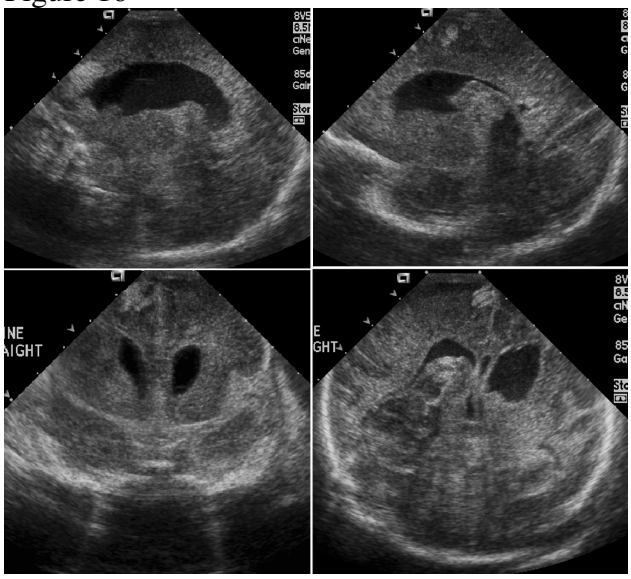

Figure 17

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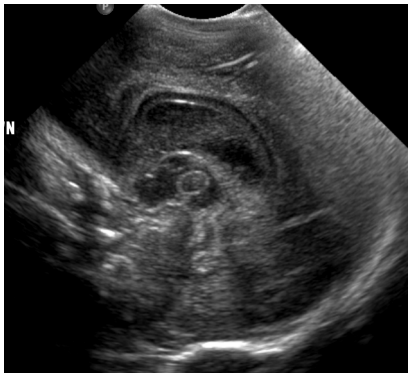

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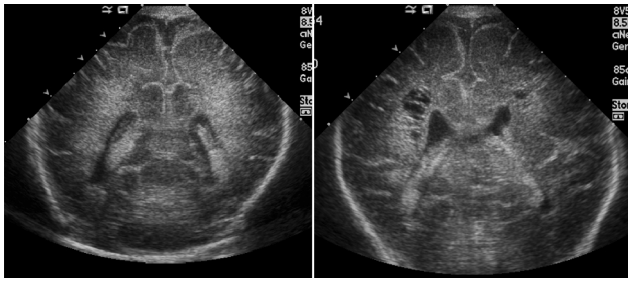

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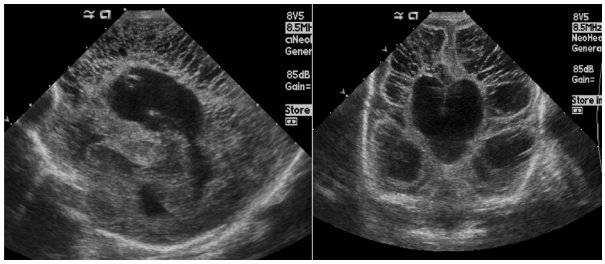

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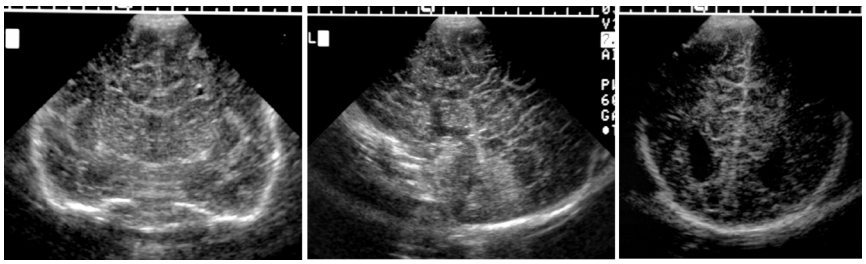

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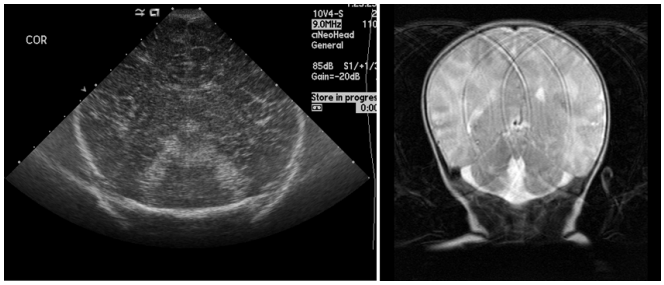

Figure 23